

Potential Reasons Why Physicians Underuse Lung-Protective Ventilation: A Retrospective Cohort Study Using Physician Documentation

Mark E Mikkelsen MD, Pali M Dedhiya MD, Ravi Kalhan MD, Robert J Gallop PhD, Paul N Lanken MD, and Barry D Fuchs MD

BACKGROUND: Physicians often fail to use lung-protective ventilation (LPV) in patients with acute lung injury. **OBJECTIVE:** To use physician documentation to identify why physicians did not initiate or continue LPV in patients with acute lung injury. **METHODS:** This was a retrospective cohort study in a university hospital. The study period was September 2000 through November 2002. In our primary analysis, LPV was defined as use of a tidal volume ≤ 7.5 mL/kg predicted body weight (PBW). We also conducted a sensitivity analysis in which we defined LPV as use of a tidal volume ≤ 6.0 mL/kg PBW. **RESULTS:** In our primary analysis, in 42 (56%) of 75 cases, physicians used or intended to use LPV. Of these 42 subjects, 12 received LPV transiently, and 6 never received LPV, despite the fact that the physician ordered or documented LPV use. In 21 of the 33 remaining cases the physicians documented concerns or clinical criteria that may explain why LPV was not used: relative contraindications to LPV ($n = 2$), change of care goal to comfort care only ($n = 1$), rapid resolution of hypoxemia ($n = 4$), and consideration of alternative diagnoses for which LPV was not indicated ($n = 14$). Of the 12 cases where LPV was used transiently, diagnostic uncertainty ($n = 6$) was a common finding. The sensitivity analysis yielded explanations in similar proportions. **CONCLUSIONS:** LPV, once initiated, is often discontinued. Uncertainty in the diagnosis of acute lung injury appears to be an important barrier to initiating and continuing LPV, whereas concerns regarding metabolic acidosis and clinical changes (hypoxemia improved) may prevent the initiation of LPV. Even when physicians believe they are using LPV, they may not be, which suggests that protocol-implementation failure is an important barrier to use of LPV. *Key words:* lung-protective ventilation, acute respiratory distress syndrome, ventilator, tidal volume, protocol, physician practice patterns. [Respir Care 2008;53(4):455–461. © 2008 Daedalus Enterprises]

Introduction

Failure to effectively implement evidence into clinical practice is one of the most important challenges in medicine.¹ Mechanical ventilation with a lung-protective ven-

tilation (LPV) strategy for patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) is no exception.

Three randomized controlled trials found that LPV strategies that limited tidal volume (V_T) and airway pressure

Mark E Mikkelsen MD, Paul N Lanken MD, and Barry D Fuchs MD are affiliated with the Division of Pulmonary, Allergy, and Critical Care; University of Pennsylvania Health System, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania. Pali M Dedhiya MD is affiliated with the Division of Pulmonary and Critical Care, Johns Hopkins Hospital, Baltimore, Maryland. Ravi Kalhan MD is affiliated with the Division of Pulmonary and Critical Care, Northwestern University Feinberg School of Medicine, Chicago, Illinois. Robert J Gallop PhD is affiliated with the Department of Mathematics, Applied Statistics Program, West Chester University of Pennsylvania, West Chester, Pennsylvania.

The study was supported in part by grant N01-HR46058, and training grant T32 HL07891, National Institutes of Health, National Heart, Lung and Blood Institute, Bethesda, Maryland.

The authors report no conflicts of interest related to the content of this paper.

Correspondence: Mark E Mikkelsen MD, Pulmonary, Allergy, and Critical Care Division, Hospital of the University of Pennsylvania, 836 W Gates Pavilion, 3400 Spruce Street, Philadelphia PA 19104. E-mail: mark.mikkelsen@uphs.upenn.edu.

were associated with lower mortality in patients with ALI.²⁻⁴ However, despite that evidence, subsequent studies found that the proportion of patients who received LPV remained modest.⁵⁻⁸ We previously found that physicians were compliant with LPV in a minority of patients, even after excluding patients for whom LPV was contraindicated at the onset of ALI.⁹

Though there have been studies of the barriers to implementing evidence-based medicine,¹⁰ only one has investigated potential reasons clinicians do not employ LPV. By surveying respiratory therapists and nurses, Rubenfeld et al identified many potential barriers to initiating and continuing LPV in patients with ALI.¹¹ However, the impressions of those practitioners may not accurately reflect the barriers experienced by physicians. In fact, the reasons physicians do not employ LPV are unknown and have not been directly investigated to date.

The primary aim of this retrospective study was to identify potential reasons physicians did not initiate or did discontinue LPV. We studied physician documentation by returning to the medical records of our previously defined cohort of ALI patients.⁹ Preliminary data from this study were reported previously in abstract form.^{12,13}

Methods

This study was approved by the University of Pennsylvania institutional review board, which waived the informed consent requirement.

Study Population: ALI Cohort

This study involved a systematic review of the medical records of a previously defined cohort of 88 patients⁹ prospectively identified as meeting American-European Consensus Conference criteria for ALI¹⁴ by a screening research coordinator and physician of the National Institutes of Health ARDS Clinical Trials Network (ARDS Network) from September 2000 (4 months after the pivotal ARDS Network study²) to November 2002. Neither the research coordinator nor the research physician were involved in direct patient care, and the physicians of record were blinded to the researchers' ALI assessments. After excluding 8 patients whose charts were missing and 5 who either expired or no longer required mechanical ventilation 48 h after ALI onset, 75 subjects were available for this study.

Definition of LPV

For consistency, we chose to employ the same definition of LPV that we used in our prior study, which was use of a $V_T \leq 7.5$ mL/kg predicted body weight (PBW) 48 h after ALI onset.⁹ This threshold was chosen because it is

approximately 1.5 standard deviations above the mean V_T used in the intervention group in the ARDS Network trial² (ie, > 93% of subjects in the LPV arm received $V_T \leq 7.5$ mL/kg PBW).² By using this more liberal definition of LPV we intended to minimize misclassification bias (ie, limit the number of cases incorrectly deemed noncompliant with LPV). LPV use was determined by whether the V_T threshold was achieved, irrespective of the mechanical ventilation mode. Admittedly, the decision to classify pressure-support ventilation V_T below the LPV threshold as compliant with LPV exposes this categorization to potential misclassification bias. We chose 48 h for the present study because it provided physicians with a reasonable amount of time to apply LPV and provided at least 2 days of physician documentation to review.

In a sensitivity analysis we reanalyzed the data with a more strict LPV definition: $V_T \leq 6.0$ mL/kg PBW 48 h after ALI onset. This criterion was used in the first published study that assessed LPV compliance.⁵ The sensitivity analysis permits comparison of results across 2 thresholds to determine how robust our findings were to our primary definition of LPV.

Case Report Form

An initial draft of the case report form was developed based on input from 3 intensivists (BDF, PNL, and Jason D Christie MD MSCE) at the Hospital of the University of Pennsylvania. After a pilot test by 2 physicians (MEM, PMD), the case report form was revised to the final 8-question case report form used for the study (Table 1). In addition to these questions, the form also included content to abstract demographic variables, respiratory mechanics, and ventilation mode, and all patient-delivered V_T values.

Retrospective Data Collection: Chart Abstraction

Two physicians (MEM, PMD), blinded to the outcome of LPV use by being blinded to height and predicted body weight, independently reviewed the 75 charts, using the case report form. Documents surveyed included physician notes, ventilator orders, and flow sheets during the initial 48 h after onset of ALI. All patient-delivered V_T values documented on the respiratory flow sheets were recorded to identify transient LPV use (eg, $V_T \leq 7.5$ mL/kg PBW any time during the initial 48 h after ALI onset, but not at 48 h). If available, the plateau pressure at 48 h was recorded. Actual body weight, if available, was also recorded from the nursing flow sheets.

The data and outcomes abstracted were based on the pre-drafted case report form. Relative contraindications to LPV (eg, severe acidosis or critical hypoxemia) were considered only if the clinicians explicitly stated that a contraindication precluded the use of LPV. A change of code

WHY PHYSICIANS UNDERUSE LUNG-PROTECTIVE VENTILATION

Table 1. Agreement Between Physician Reviewers Who Used the Pre-Drafted Case Report Form, by Question

Question	Interclass Correlation	95% CI
1. Any documentation that LPV was attempted or ordered?	1	NA
2. Is there a relative contraindication (cardiovascular dysfunction or respiratory/metabolic dysfunction) for LPV explicitly stated in the chart?	1	NA
3. Is there an absolute contraindication (eg, pregnancy, elevated intracranial pressure, sickle cell crisis) for LPV explicitly stated in the chart?	1	NA
4. Is there documentation that LPV was deemed not applicable (comfort care, rapidly improving)?	0.923	0.862–0.942
5. Is there any other reason stated for not using LPV (eg, patient is too well, general appearance, oxygenating too well, concern for sedation while on pressure support)?	0.815	0.693–0.865
6. An alternative diagnosis for the acute bilateral infiltrates is written by the physician that is not consistent with or a cause of ALI (eg, congestive heart failure, diffuse alveolar hemorrhage, chronic infiltrates)?	0.962	0.932–0.972
7. Does the physician question the presence of bilateral infiltrates on chest radiograph (eg, absence of bilateral pulmonary infiltrates, infiltrates are chronic, unilateral or bilateral atelectasis, mass, or effusions)?	0.910	0.849–0.937
8. Final chart diagnosis for bilateral infiltrates (ALI or diagnosis consistent with ALI vs congestive heart failure/ volume overload vs diffuse alveolar hemorrhage vs other vs none found in chart)	0.982	0.969–0.987

CI = confidence interval

LPV = lung-protective ventilation (tidal volume \leq 7.5 mL/kg predicted body weight)

NA = not applicable

status to comfort care only was considered a change in clinical status that obviated LPV. Rapid clinical improvement in hypoxemia was defined as a \geq 50% relative decrease in fraction of inspired oxygen within 24 hours of ALI diagnosis, without an accompanying increase in positive end-expiratory pressure to $>$ 7.5 cm H₂O. We took that level of positive end-expiratory pressure into consideration to minimize the potential for misclassification bias in patients with whom the physicians preferentially adjusted positive end-expiratory pressure to combat hypoxemia. To determine the final diagnosis for respiratory failure documented by the physician, the reviewers surveyed the physician notes from the 7 days following ALI onset.

Statistical Analysis

All statistical analyses were performed with statistics software (SAS 9.1, Cary, North Carolina). The chi-square statistic was used to compare binomial proportions between groups. To assess agreement between the physician reviewers we used the interclass correlation coefficient, which is a continuous analog of the kappa statistic, which is a better index of agreement when several of the questions have more than 3 potential responses. The interclass correlation coefficient statistic ranges from 0 to 1: a value

below 0.4 indicates poor reliability; 0.5–0.7 indicates minimal acceptable reliability; and a value above 0.75 indicates excellent reliability.^{15,16}

Results

Agreement between the 2 physician reviewers was excellent; the mean interclass correlation coefficient was 0.949 (range 0.82–1.00 for each question) (see Table 1). Table 2 shows the pertinent patient characteristics. Table 3 shows the use of and reasons for non-use of LPV in the primary analysis. Forty-eight percent of the patients (36 of 75) were on LPV for some period of time during the initial 48 h of ALI, but only 24 patients (32%) had LPV initiated and sustained through the 48-hour period. Twelve of 75 patients (16%) were on LPV transiently, 7 of whom were on pressure-support ventilation at their V_T nadir. Six patients (8%) never received LPV despite the fact that the physicians either ordered the protocol for LPV ($n = 1$), documented that they were using LPV ($n = 4$), or both ($n = 1$).

Of the 33 cases where there was no documentation of use or intent to use LPV, the physicians documented concerns or clinical criteria that may explain why LPV was not used in 21 cases. In 2 patients (2.7%) the physicians

Table 2. Patient Characteristics (*n* = 75)

Age (mean y)	50
APACHE III score (mean)	76
30-day mortality (%)	38
Male (%)	71
MICU (%)	41
SICU (%)	59
Caucasian (%)	62
African-American (%)	38
Final diagnosis (%)	
Sepsis	31
Pneumonia	15
Aspiration	19
Congestive heart failure	9
Trauma	9
ARDS	8
No diagnosis	9
Median V_T at 48 h (mL/kg PBW and IQR)	8.36 (7.29–9.4)
Median V_T (nadir) during initial 48 h (mL/kg PBW and IQR)	7.72 (5.99–8.79)

APACHE = Acute Physiology and Chronic Health Evaluation
 MICU = medical intensive care unit
 SICU = surgical intensive care unit
 ARDS = acute respiratory distress syndrome
 V_T = tidal volume
 PBW = predicted body weight
 IQR = interquartile range

Table 3. LPV Use (*n* = 75)

LPV sustained (on LPV at 48 h) (%)	32
LPV used transiently (%)	16
Reason LPV not used (%)	
Diagnostic uncertainty	18.7
No documented explanation	16
Implementation failure	8
Change in clinical status*	6.7
Relative contraindication (metabolic acidosis)	2.7

* Includes 4 patients who rapidly improved and 1 patient who transitioned to comfort care
 LPV = lung-protective ventilation (tidal volume \leq 7.5 mL/kg of predicted body weight)

documented that they were not using LPV because of a relative contraindication (metabolic acidosis, which prompted the physician to “hyperventilate [to compensate] for acidosis” [pH 7.06, HCO₃ 11 mEq/L] or “adjust V_T for pH” [pH 7.10, HCO₃ 16 mEq/L]). In another 5 patients (6.7%) the physicians documented rapid resolution of hypoxemia (*n* = 4) or change of the care goal to comfort only (*n* = 1). In another 14 patients (18.7%) there was documentation that the physicians considered diagnoses other than ALI: different interpretation of the chest radiograph, such as atelectasis alone (*n* = 2); alternative etiology for the bilateral infiltrates, such as congestive heart

failure (*n* = 11); or lymphoma (*n* = 1). In 11 of these 14 cases the physician documented a final diagnosis consistent with ALI (final diagnosis congestive heart failure, *n* = 3). Plateau pressure during the second day was documented in 22 of the 33 cases that did not have LPV ordered. The day-2 plateau pressure was \leq 30 cm H₂O in 18 of the 22 cases (81.8%).

Actual body weight was available in only 8 of the 33 cases where there was no documentation of use or intent to use LPV. On average, actual body weight was 29.7% greater than PBW, based on the subject’s gender and height. Of the 8 cases where actual body weight was recorded, half of the cases were on a $V_T \leq$ 7.5 mL/kg actual body weight, rather than PBW, and sustained below that threshold until 48 h after ALI onset, and 5 of the 8 patients were on a $V_T \leq$ 7.5 mL/kg actual body weight at some point during the initial 48 h but that V_T was not sustained.

Of the 12 patients transiently on LPV in the primary analysis, potential explanations documented for discontinuing LPV included rapid resolution of hypoxemia (*n* = 1), changed to comfort care only (*n* = 1), consideration of diagnoses other than ALI (*n* = 6, including a different interpretation of the chest radiograph, *n* = 2), and an alternative etiology for the bilateral infiltrates (congestive heart failure, *n* = 4). Eight of these 12 patients had a day-2 plateau pressure recorded: 6 of the 8 had a plateau pressure \leq 30 cm H₂O. All 12 patients had a final diagnosis consistent with ALI.

By our primary definition of LPV, LPV was underutilized during the study period. In 2000, 5 of 13 subjects (38.5%) were on LPV. In 2001, 6 of 26 subjects (23.1%) were on LPV. In 2002, 13 of 36 subjects (36.1%) were on LPV. Those proportions of LPV use were not significantly different from one another (*p* > 0.20 for each comparison).

The sensitivity analysis, in which we redefined LPV as a measured $V_T \leq$ 6.0 mL/kg PBW, yielded similar proportions (not significantly different) in regard to potential reasons patients did not receive LPV (Table 4). Post hoc analysis indicated that patients were more likely to have LPV sustained through to 48 h at the less strict definition of LPV (odds ratio 3.42, 95% confidence interval 1.09–10.72, *p* = 0.03).

Discussion

Many hypotheses have been proposed to explain why physicians under-use LPV in patients with ALI. Although we, and others, have previously reported clinical data that may differentiate patients who fail to receive LPV, no study has investigated ordering physicians directly to determine why LPV was not used. Given the potential for physician response bias in prospective surveys of physician practice patterns (ie, repetitive survey encounters

WHY PHYSICIANS UNDERUSE LUNG-PROTECTIVE VENTILATION

Table 4. Use of LPV and Potential Reasons LPV Was Not Used, by Different Definitions of LPV

	Primary Analysis ($V_T \leq 7.5$ mL/kg PBW) <i>n</i> (%)	Sensitivity Analysis ($V_T \leq 6.0$ mL/kg PBW) <i>n</i> (%)
LPV sustained (on LPV at 48 h)	24 (32)	7 (9.3)
LPV used transiently	12 (16)	12 (16)
LPV intended, but implementation failed	6 (8)	9 (12)
Relative contraindication (metabolic acidosis)	2 (2.7)	3 (4)
Change in clinical status (patient improved rapidly or transitioned to comfort care)	5 (6.7)	7 (9.3)
Diagnostic uncertainty	14 (18.7)	21 (28)
No documented explanation for why LPV was not used*	12 (16)	16 (21.3)

* In the sensitivity analysis, day-2 plateau pressure was ≤ 30 cm H₂O in 4 of the 8 cases in which it was recorded.

LPV = lung-protective ventilation

V_T = tidal volume

PBW = predicted body weight

would probably alter physician responses [the Hawthorne effect]) and evidence that disagreement exists between physicians' actual behavior and their perception of their behavior,^{17,18} as a first approach we returned to the medical record notations of the ordering physicians of our previously established cohort of ALI patients to determine why LPV was not used.

As we found in our initial report, approximately one third of the patients were on LPV at 48 h. What is novel in our present study is how often we found that physicians used LPV transiently or documented that they were using LPV even though they were not. And in the remaining cases we found clinical explanations for why LPV may not have been used in nearly two thirds of the cohort, and noted that uncertainty about the diagnosis of ALI appears to be an important barrier to initiating and continuing LPV.

All prior studies that assessed LPV compliance in patients with ALI used a single V_T measurement on one⁶ or several^{5,9} days to assess physician practice. In the present study we reviewed all the V_T used during the initial 48 h of ALI to identify cases where physicians used LPV, and we captured an additional 12 patients (a 50% increase) who were on LPV transiently. Although transient use may still be considered "noncompliance" if ALI persists and, as we acknowledge, our estimate may be inflated due to misclassification bias, this distinction is important and necessary when investigating the causes of "translation failure" (ie, barriers to initiating versus barriers to continuing LPV). By measuring only one or several V_T to determine the use of LPV, prior estimates of LPV use may not have provided an accurate assessment of its actual use in clinical practice.

A previously unrecognized barrier to LPV use that emerged from our study was the failure to effectively implement the LPV protocol despite a physician's perceived order. Although physician-specific issues (documenting, but not ordering the protocol) and system malfunctions

(eg, computer order entry transmittals and communication between health-care providers) may explain these findings, an alternative explanation suggested by our data is that the actual rather than predicted body weight may have been used to determine the V_T , as Young et al⁶ and Rubenfeld et al suggested.¹¹ A consolidated LPV order set was available at our institution at the time of this study, and it required the entry of the patient's height to calculate predicted body weight, but the LPV order set was not required to enter the ventilator settings and was, in fact, infrequently used.

In a significant proportion of patients the physicians documented either a change in clinical status, which they thought obviated LPV, or a fear that low V_T would adversely affect patient safety. Though some of these explanations have been previously hypothesized (eg, concerns regarding acidosis, based on the survey study by Rubenfeld et al¹¹), others had not been considered or investigated (eg, rapid resolution of hypoxemia, transition to comfort care only). Furthermore, our findings suggest that physician uncertainty about the diagnosis of ALI is an important barrier to LPV use. Though previous studies have suggested that physicians fail to recognize ALI,^{6,10} the observation that diagnostic uncertainty is relatively common is one plausible explanation for non-use of LPV. Although several studies have shown significant variability, even among experts, in the interpretation of the chest radiographs of ALI patients,^{19,20} others have demonstrated the predictive accuracy of applying ALI criteria within 24 hours of ALI onset.²¹ In view of data that suggest the utility of LPV in patients at risk for ALI,^{22,23} and our findings that nearly all of these patients with uncertain diagnoses were eventually ascribed a diagnosis consistent with ALI by their physician, it seems appropriate to initiate LPV in all patients who meet ALI criteria, even when competing diagnoses are being considered.

Although not explicitly documented as a reason for not using LPV when available, 82% of the patients who never received LPV in the primary analysis had a plateau pressure ≤ 30 cm H₂O 48 h after ALI onset. Although recent evidence suggests that reducing V_T in patients with plateau pressure ≤ 30 cm H₂O is associated with a survival benefit,²⁴ it is possible that clinicians, at the time of our study, disregarded the importance of reducing V_T when airway pressure was at goal. This potential barrier to LPV implementation, which warrants further investigation, should be studied prospectively.

When we compared the sensitivity analysis to the primary analysis we found that, once initiated, continuing LPV was significantly less likely at the more strict definition of LPV. This suggests that clinicians may experience more difficulty sustaining lower V_T , albeit for unclear reasons. Also, protocol implementation failure (ie, the physician was under the mistaken impression that the patient was on LPV) and diagnostic uncertainty emerged as potential barriers to LPV use with both LPV definitions.

Only one study has investigated why clinicians underutilize LPV.¹¹ Note that our study of physician documentation did not confirm several of the perceptions of critical care nurses and respiratory therapists noted in the survey study by Rubenfeld et al,¹¹ namely that patient discomfort, tachypnea, hypercapnic acidosis, and worsening oxygenation were important barriers to continuing LPV.¹¹ However, our study does not refute those findings either, so they remain potential reasons why physicians might discontinue LPV. In fact, it is possible that physicians did perceive these factors as barriers, which could explain our finding that clinicians experience more difficulty sustaining lower V_T , but they either failed to document these concerns or our methods of analysis failed to capture them.

There are several potential limitations to our study. First, documentation does not equate to clinician cognition. Our deductions regarding physicians' reasons and reasoning were based on what was documented in the daily progress notes. The validity of physician documentation in the medical record has been demonstrated in other realms of medical practice, such as in cerebrovascular research and quality of care assessments,^{25–28} and is commonly used in medical malpractice defense, fee abstraction, and illness-severity assessments. How valid physician documentation is in the critical care setting is unknown. Given the dynamic nature of critical care medicine, it is reasonable to assume that certain medical decisions would go undocumented, as observed in other fields,²⁸ which would limit the value of using the medical record to ascertain clinician cognition. However, that limitation should not undermine the validity of an observed documentation, so we believe that physician documentation provides useful information as a first approach to understanding why physicians underuse LPV. Future research should investigate the validity of

using the medical record from critical care settings, and our primary aim should be reassessed with prospective surveys of physicians, respiratory therapists, and critical care nurses, albeit with the limitations already discussed.

Second, because our study was performed in a single academic, ARDS-Network-affiliated institution, our findings may not be generalizable to physicians elsewhere (eg, community-based hospitals).

Third, our cohort is dated by several years. Nevertheless, recent publications on this subject, the most recent of which was published in July 2007, demonstrate that the problem persists.^{5–9} Future endeavors should investigate why physicians continue to under-use LPV, taking into consideration the potential reasons that we identified, and determine whether alternative ventilation modes (eg, airway pressure-release ventilation, bi-level positive airway pressure) are ameliorating or exacerbating the problem.

Conclusions

By reviewing all V_T used over the initial, critical hours after ALI onset, we found that the majority of patients never received LPV at any point during the first 48 h of ALI, and that LPV, once initiated, is often discontinued. After ascertaining the transient use of LPV, and recognizing that the intent to use LPV does not always lead to implementation, we studied physicians' documentation to determine why LPV was not initiated or discontinued in patients who met ALI criteria. Our study, the first to directly assess why physicians do not use LPV, suggests that uncertainty in making the diagnosis of ALI may be an important barrier to implementing and continuing LPV, and that physicians may prioritize limiting airway pressure to limiting V_T .

ACKNOWLEDGMENTS

We thank Jason D Christie MD MSCE and Theodore J Iwashyna MD PhD, Pulmonary, Allergy and Critical Care Division, Hospital of the University of Pennsylvania, for their assistance in the design of the study and editing of the manuscript.

REFERENCES

1. Institute of Medicine Committee on Quality of Health Care in America. Crossing the quality chasm: a new health system for the 21st century. Washington DC: National Academy Press, 2001.
2. The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000;342(18):1301–1308.
3. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, et al. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. *N Engl J Med* 1998;338(6):347–354.
4. Villar J, Kacmarek RM, Pérez-Méndez L, Aguirre-Jame A. A high positive end-expiratory pressure, low tidal volume ventilatory strategy improves outcome in persistent acute respiratory distress syn-

- drome: a randomized, controlled trial. *Crit Care Med* 2006;34(5):1311–1318.
5. Rubenfeld GD, Caldwell E, Hudson L. Publication of study results does not increase use of lung protective ventilation in patients with acute lung injury (abstract). *Am J Respir Crit Care Med* 2001;163:A295
 6. Young MP, Manning HL, Wilson DL, Mette SA, Riker RR, Leiter JC, et al. Ventilation of patients with acute lung injury and acute respiratory distress syndrome: Has new evidence changed clinical practice? *Crit Care Med* 2004;32(6):1260–1265.
 7. Weinert CR, Gross CR, Marinelli WA. Impact of randomized trial results on acute lung injury ventilator therapy in teaching hospitals. *Am J Respir Crit Care Med* 2003;167(10):1304–1309.
 8. Ilan R, Fowler RA, Geerts R, Pinto R, Sibbald WJ, Martin CM. Knowledge translation in critical care: Factors associated with prescription of commonly recommended best practices for critically ill patients. *Crit Care Med* 2007;35(7):1696–1702.
 9. Kalhan R, Mikkelsen M, Dedhiya P, Christie J, Gaughan C, Lanken PN, et al. Underuse of lung protective ventilation: analysis of potential factors to explain physician behavior. *Crit Care Med* 2006;34(2):300–306.
 10. Cabana MD, Rand CS, Powe NR, Wu AW, Wilson MH, Abboud PA, Rubin HR. Why don't physicians follow clinical practice guidelines? A framework for improvement. *JAMA* 1999;282(15):1458–1465.
 11. Rubenfeld GD, Cooper C, Carter G, Thompson B, Hudson LD. Barriers to providing lung protective ventilation to patients with acute lung injury. *Crit Care Med* 2004;32(6):1289–1293.
 12. Dedhiya P, Mikkelsen ME, Kalhan R, et al. Early clinician recognition of acute lung injury may increase use of lung protective ventilation (abstract). *Am J Respir Crit Care Med* 2004;169(Suppl):A17.
 13. Mikkelsen ME, Dedhiya P, Kalhan R. Factors contributing to physician non-compliance in use of lung protective ventilation in patients with acute lung injury (abstract). *Am J Respir Crit Care Med* 2004;169(Suppl):A17.
 14. Bernard GR, Artigas A, Brigham KL, Carlet J, Falke K, Hudson L, et al. The American-European consensus conference on ARDS. Definitions, mechanisms, relevant outcomes, and clinical trial coordination. *Am J Respir Crit Care Med* 1994;149(3 Pt 1):818–824.
 15. Shrout PE, Fleiss JL. Intraclass correlations: use in assessing rater reliability. *Psychological Bulletin* 1979;86:420–428.
 16. Streiner DL, Norman G. Health measurement scales: a practical guide to their development and use. 3rd edition. Oxford: Oxford University Press, 2003.
 17. Leaf DA, Neighbor WE, Schaad D, Scott CS. A comparison of self-report and chart audit in studying resident physician assessment of cardiac risk factors. *J Gen Intern Med* 1995;10(4):194–198.
 18. Montañó DE, Phillips WR. Cancer screening by primary care physicians: a comparison of rates obtained from physician self-report, patient survey, and chart audit. *Am J Public Health* 1995;85(6):795–800.
 19. Rubenfeld GD, Caldwell E, Granton J, Hudson LD, Matthay MA. Interobserver variability in applying a radiographic definition of ARDS. *Chest* 1999;116(5):1347–1353.
 20. Meade MO, Cook RJ, Guyatt GH, Groll R, Kachura JR, Bedard M, et al. Inter-observer variation in interpreting chest radiographs for the diagnosis of acute respiratory distress syndrome. *Am J Respir Crit Care Med* 2000;161(1):85–90.
 21. Gajic O, Dara SI, Mendez JL, Adesanya AO, Festic E, Caples SM, et al. Ventilator-associated lung injury in patients without acute lung injury at the onset of mechanical ventilation. *Crit Care Med* 2004;32(9):1817–1824.
 22. Gajic O, Frutos-Vivar F, Esteban A, Hubmayr RD, Anzueto A. Ventilator settings as a risk factor for acute respiratory distress syndrome in mechanically ventilated patients. *Intensive Care Med* 2005;31(7):922–926.
 23. Yilmaz M, Keegan MT, Iscimen R, Afessa B, Buck CF, Hubmayr RD, Gajic O. Toward the prevention of acute lung injury: protocol-guided limitation of large tidal volume ventilation and inappropriate transfusion. *Crit Care Med* 2007;35(7):1660–1666.
 24. Brower R, Krishnan J, Thompson BT, et al. Effects of tidal volume reduction in acute lung injury patients with inspiratory plateau pressures < 32 cm H₂O before tidal volume reduction (abstract). *Am J Respir Crit Care Med* 2003;167(Suppl):A616.
 25. Kasner SE, Chalela JA, Luciano JM, Cucchiara BL, Raps EC, McGarvey ML, et al. Reliability and validity of estimating the NIH stroke scale score from medical records. *Stroke* 1999;30(8):1534–1537.
 26. Williams LS, Engin YY, Lopez-Yunez AM. Retrospective assessment of initial stroke severity with the NIH stroke scale. *Stroke* 2000;31(4):858–862.
 27. Stange KC, Zyzanski SJ, Smith TF, Kelly R, Langa DM, Flocke SA, Jaén CR. How valid are medical records and patient questionnaires for physician profiling and health services research? A comparison with direct observation of patient visits. *Med Care* 1998;36(6):851–867.
 28. Audet AM, Goodnough LT, Parvin CA. Evaluating the appropriateness of red blood cell transfusions: the limitations of retrospective medical record reviews. *J Qual Health Care* 1996;8(1):41–49.